

ENDOTRACHEAL TUBE CUFF LEAK WITH MYSTERIOUS LARYNGOTRACHEAL PATHOLOGY

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Abstract

A 63 year-old obese man with gastroesophageal reflux disease, hiatal hernia, and no known history of airway pathology was to undergo a total knee arthroplasty. After intubation, however, repeated cuff leaks, decreasing tidal volumes, and desaturations prompted five additional endotracheal tube placements. Findings on radiography, computed tomography, and fiberoptic laryngoscopy and tracheoscopy were equivocal. Factors contributing to this challenge of persistent and repeated cuff leaks in the absence of known airway pathology could include various laryngotracheal abnormalities.

Keywords include endotracheal tube cuff, tracheal stenosis, and tracheomalacia

Case Description

A 63 year-old man suffered from chronic left knee osteoarthritis secondary to remote trauma. After unsuccessful medical management and multiple knee surgeries, a total knee arthroplasty was planned. His medical history included gastroesophageal reflux disease (GERD), hiatal hernia, dyslipidemia, and benign prostatic hypertrophy. His surgical history included multiple left knee surgeries, right knee arthroscopy, lumbar discectomy, and lipoma excision. He denied any previous anesthetic complications, intensive care unit admissions, or prolonged periods of endotracheal intubation or mechanical ventilation. His daily medications included fenofibrate, atorvastatin, tamsulosin, esomeprazole, and aspirin. His exercise tolerance was severely limited by knee pain, and he was unable to walk 1 block.

Upon physical examination his weight was 125 kg (276 lb), height 193 cm (6 ft 4 in), and body mass index (BMI) was 33.6 (obese). Airway evaluation revealed a Mallampati Class II, beard, adequate grade I cervical mobility, normal Wilson's (upper lip bite test) position A, intact dentition, and moderate micrognathia. His airway distances were within normal limits: inter-incisors 5 cm, thyromental 12 cm, sternomental 22 cm, neck circumference 36 cm^{1,2,3,4}. His renal and liver function tests, complete blood count, coagulation studies, electrocardiogram, chest radiograph, and adenosine nuclear cardiac study were all within normal limits.

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On the day of surgery, the patient was premedicated with midazolam 2 mg, and monitored with standard ASA monitors, including noninvasive blood pressure, temperature, end-tidal carbon dioxide (ETCO₂), electrocardiogram, and pulse oximeter in the operating room. The blood pressure was 159/94, the heart rate was 62 beats per minute (BPM), and the oxygen saturation (SpO₂) was 100%. The patient was preoxygenated with 100% oxygen at 10 liters per min (L/min) for three minutes. A rapid sequence induction was planned for frequent GERD symptoms and a history of hiatal hernia, which were controlled on esomeprazole. General anesthesia was induced with lidocaine 50 mg, fentanyl 150 mcg, propofol 200 mg, and succinylcholine 100 mg. After one failed intubation attempt by a medical student, the patient was tracheally intubated using a Macintosh 4 laryngoscope. A grade 2a view was achieved with cricoid pressure.

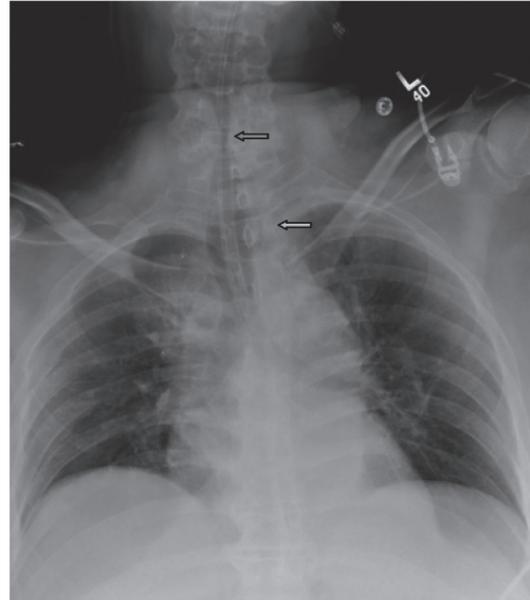
The 8.0 endotracheal tube (ETT) cuff was inserted, inflated, ETCO₂ was detected, and breath sounds were bilateral and equal upon auscultation. The ETT was secured at 24 cm at the lip, no leak was present at 30 cm H₂O, and the SpO₂ was 100%. Then, cisatracurium 10 mg was administered, end-tidal sevoflurane was increased to 2%, and the fraction of inspired oxygen (FiO₂) was decreased to 50% at 2 L/min. Vital signs, capnogram, ETCO₂, tidal volumes, and peak inspiratory pressures were normal for ten minutes. Subsequently, tidal volumes decreased from 700 cc to 400-500 cc, the SpO₂ decreased to the low 90s, and an ETT cuff leak was apparent at less than 20 cm H₂O after an additional 10-20 cc of injected air.

Five subsequent ETT placements were performed (Table 1). After each successful ETT placement, the cuffs were inflated, breath sounds were bilateral, and no leak was present at 30 cm H₂O. A period of normal vital signs, capnogram, ETCO₂ tidal volumes, and peak inspiratory pressures followed. Afterwards, tidal volumes decreased from 700 cc to 400-500 cc, the SpO₂ decreased to the low 90s, and an ETT cuff leak was apparent at less than 20 cm H₂O after 10-20 cc of injected air. All cuffs were confirmed as functional prior to intubation. Except for the final ETT, each tube was unable to be advanced past 24 cm. The final successful ETT was secured at 26 cm.

An intraoperative chest radiograph (Fig. 1) and

Fig. 1

Red arrow, tracheal stenosis; Green arrow, post-stenotic tracheal dilation



a lateral cervical spine radiograph were then ordered (Table 2), and otolaryngology (ENT) was consulted for rigid bronchoscopic evaluation to rule out tracheal stenosis, mass, or rupture. The patient was given additional muscle relaxant, and rigid laryngoscopy and tracheoscopy (Video 1-3) were then performed by ENT with intermittent ventilation using a 6.0 ETT. The patient was then extubated successfully in the operating room and taken to the post-anesthesia care unit. The

Fig. 2

Red arrow, tracheal stenosis; Green arrow, post-stenotic tracheal dilation

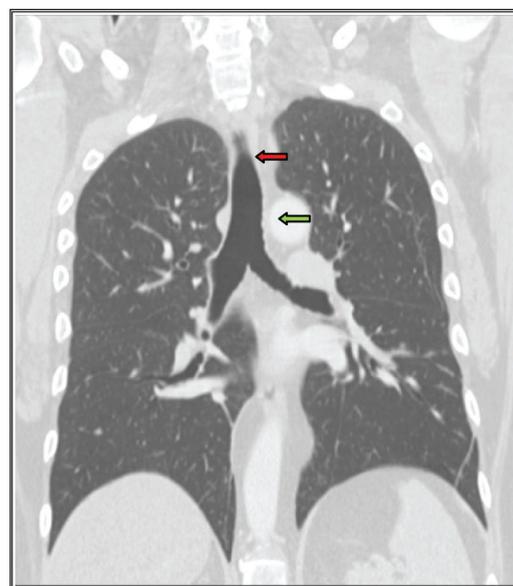


Table 1:
Endotracheal Tube Placements

ETT (mm ID)	Intubation Technique	Depth (cm)	Sustained function (min)	Comments
8.0	DL, Mac 4	24	10	Leak after OGT attempted x 3; cuff not visible on DL
8.0	Cook	24	5	Advancement past 24 cm unsuccessful
7.0	Cook	24	4	Flexible FOB view through ETT showed carina
7.0	Cook	24	2	Case cancelled after development of cuff leak
6.0	DL, Mil 3	24	2	Performed by ENT
6.0	Cook	26	>120	Flexible FOB view through ETT showed carina

DL, direct laryngoscopy; Mac 4, Macintosh 4 laryngoscope; OGT, orogastric tube; Cook, Cook airway exchange catheter; FOB, flexible bronchoscope; ETT, endotracheal tube; Mil 3, Miller 3 laryngoscope; ENT, otolaryngologist.

patient was admitted to the surgical intermediate unit, and his hospital course was uncomplicated. He denied dyspnea or hoarseness, and auscultation revealed clear lungs without stridor or wheezing. Computed tomography (CT) of the chest (Fig. 2) was ordered to further characterize chest radiograph findings.

A total of six ETT were used in this case including two 8.0, two 7.0, and two 6.0 tubes. The cuff viability of one 7.0 ETT was unable to be assessed as its pilot balloon inflating tube was torn. One 6.0 ETT was not recovered postoperatively. Testing of the remaining four cuffs by saline injection showed that both 8.0 and one 6.0 cuffs were intact, and one 7.0 cuff was ruptured. Thus, out of the six cuffs, there were three intact cuffs, one ruptured cuff, and two cuffs which were unable to be assessed.

Discussion

The present case describes a patient with repeated cuff leaks from multiple ETTs with an uncertain explanation. One possible reason for the repeated cuff leaks is tracheal stenosis which appears on chest radiograph and chest CT. The first five ETT were unable to be advanced past 24 cm, which may have been a region of subglottic stenosis. After the cuffs were inflated, there was gradual supraglottic herniation with a resulting loss of tidal volume, desaturation, and cuff leak. Further inflation then led to cephalad migration of the cuff and ETT which impeded ventilation and oxygenation. The successful, sustained placement of a 6.0 ETT at 26 cm occurred as the smaller diameter

permitted passage distal to the stenosis preventing supraglottic herniation. Our serial ETT dilations may provide a reason for both our eventual sustained ability to ventilate the patient with a sufficient seal, and for the absence of stenosis on endoscopy. In part, our attempts to secure the airway of this patient may have been curative of the patient's stenosis.

Over time, ulcerative tracheal lesions, which can be formed from a period of hypoperfusion as a result of an overinflated ETT cuff, may be replaced by membranes or webs⁵. This may represent another explanation in this case. The membrane or web would have not allowed for advancement of the ETT past 24 cm. The successful, sustained placement of the 6.0 ETT at 26 cm was possible only after perforation of the membrane or web. Subsequent tracheoscopy may have failed to visualize the membrane or web as it would have been obliterated by serial intubations or by the removal of the final ETT by ENT with the cuff fully inflated.

Ideally, the ETT should be advanced 2 cm after the cuff completely passes through the cords, or when the external tube markings are 22 to 24 cm at the lower incisors. This places the tip of the tube 2 to 4 cm above the carina in the midtrachea. After ETT placement, the cuff should be inflated to 22 – 32 cm H₂O (generally should not exceed 25 cm H₂O) until adequacy of ventilation is demonstrated. In the absence of a cuff pressure gauge, moderate tension should be felt in the pilot balloon⁵.

The majority of stenosis occurs at the cuff site, although it can also happen at the ETT tip site⁵.

Table 2:
Imaging Results

Imaging	Results
Lateral Cervical Spine	Nondiagnostic
Chest X-Ray	<u>Report 1</u>) Mass extending out of the superior mediastinum or in the lungs in the right upper lobe cannot be excluded. <u>Report 2</u>) Possible proximal tracheal stenosis, possible distal tracheal dilation, and possible right upper lobe mass.
Rigid Laryngoscopy and Tracheoscopy	Normal from the supraglottis to the main bronchi except for an enlarged trachea in the steep slope of the trachea.
Chest Computed Tomography	<u>Report 1</u>) No mediastinal mass. Small hiatal hernia. (No mention of airway.) <u>Report 2</u>) Vocal cord thickening, possible edema or hematoma, cannot rule out laryngeal mass, no intrathoracic mass. Possible proximal tracheal stenosis, possible distal tracheal dilation, possible tracheomalacia, possible tracheal membrane or web.

Excessive mucosal pressure is the principal cause of post-intubation ETT cuff site induced tracheal stenosis⁶. When the cuff pressure is greater than 30 mm Hg, it can exceed capillary perfusion pressure⁷. This can lead to mucosal edema and ischemia, which can ultimately progress to inflammation, desquamation, ulceration, and necrosis of the trachea^{5,6,7}. In hypotensive patients, capillary perfusion pressure is more easily overcome, and ulceration occurs at even lower cuff pressures^{8,9}. Resulting circumferential lesions heal by fibrosis and contraction, which can eventually lead to tracheal stenosis. Using a cuff with high volume, low pressure, and a larger contact area minimizes mucosal pressure and decreases the incidence of post-intubation tracheal stenosis¹⁰. However, the benefits of using such high volume, low pressure cuffs can be negated by cuff overinflation.

Symptoms of tracheal stenosis include dyspnea, stridor, and wheezing⁶. Upper airway obstruction classically presents with monophonic wheezing, whereas lower, small airway obstruction (asthma, COPD) is classically polyphonic wheezing. Post-intubation tracheal stenosis commonly misdiagnosed as asthma or COPD in up to 44% of patients. Symptoms may not manifest at rest until the trachea lumen has decreased to 30% of its original size which may take up to 3 months. A diagnosis of tracheal stenosis should be considered in patients with progressive dyspnea and wheezing which is unresponsive to bronchodilators, or in patients with a recent or prolonged period of endotracheal intubation. Confirmation can be obtained with radiography, computed tomography, or

endoscopy. Treatment options include steroids and surgery for symptomatic lesions. Rigid bronchoscopy and tracheal dilation with possible stent placement is the definitive treatment for mild to moderate lesions, or can be used as a temporizing measure for more severe lesions. Tracheal reconstruction is considered for severe lesions when the lumen diameter is 4 to 5 mm^{6,11}.

Another possible explanation for the persistent cuff leak is tracheomalacia and dilation, as the increased tracheal compliance and diameter did not allow for an effective cuff seal. Also, luminal narrowing from concomitant tracheal collapse may have obstructed ETT advancement past 24 cm. During the chest radiograph with positive pressure ventilation and the chest CT with spontaneous ventilation, there was evidence of tracheomalacia and dilation. Conversely, during endoscopy when the patient was paralyzed and apneic, there was no airway pressure gradient and thus no visible tracheal collapse. The staff radiologists suggested live fluoroscopy and rigid or flexible FOB without paralysis to identify possible dynamic tracheal compression during spontaneous ventilation. Lastly, the rigid bronchoscope may have masked any tracheal collapse by altering the mechanical integrity of the trachea. The endoscopy did, however, provide evidence of tracheal dilation.

Tracheomalacia is abnormal tracheal flaccidity resulting in dynamic airway collapse when extraluminal pressure is greater than intraluminal pressure, such as during expiration or coughing⁵. Tracheal cartilage erosion from previous ischemic

insult can lead to tracheomalacia⁵. The cartilage softens and the posterior membranous wall widens with possible anterior ballooning into the lumen. The average female tracheal diameter is approximately 2 cm, while the average male tracheal diameter is 2.4 cm¹². Whenever increasing cuff volumes are necessary to sustain a seal, tracheomalacia and tracheal dilation should be considered⁹. Symptoms include dyspnea, cough, wheezing, dysphagia, and recurrent respiratory infections. Difficulty weaning from mechanical ventilation or repeated failed extubations may also indicate functional tracheal collapse. Short term treatment may include non-invasive positive pressure ventilation such as CPAP or BIPAP⁵.

In summary, the present case describes a patient

with no known history of airway pathology that required five additional endotracheal tube placements. Factors contributing to this challenge of persistent and repeated cuff leaks in the absence of known airway pathology could include various laryngotracheal abnormalities. Clinical anesthesiologists must appreciate the differential diagnosis and potential treatment plan when confronted with such a challenging problem.

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